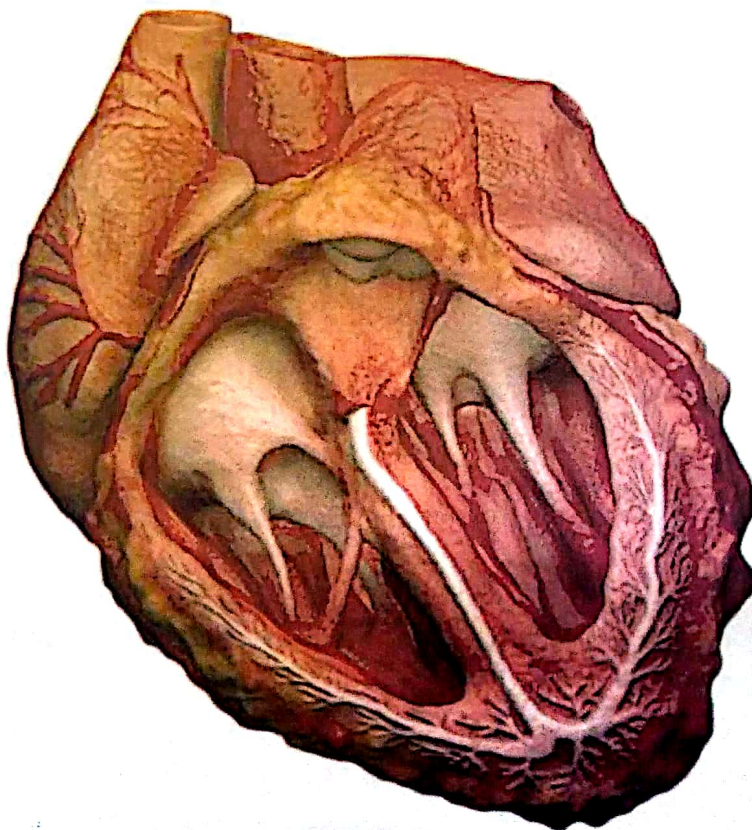


Easy Pathology

— Dr. Abdelrahman Khalifa —

Cardiovascular System

Part 1



VISIT...

LANZAROTE
Caliente.COM

Rheumatic fever

95% in child other 6 congenital

Autoimmune collagen disease, following streptococcal infection, affecting multiple systems

تبعي في اكثر من اعضاء

Predisposing factors: Children 5-15y. سن 5-15

- URT infection by group A Beta-hemolytic strept., e.g. tonsillitis (low immunity - crowding)
- genetic factor

Pathogenesis:

Anti streptolysin O

- After 2-3 weeks of URTI by group A bet hem strept. → elevated ASO antibody

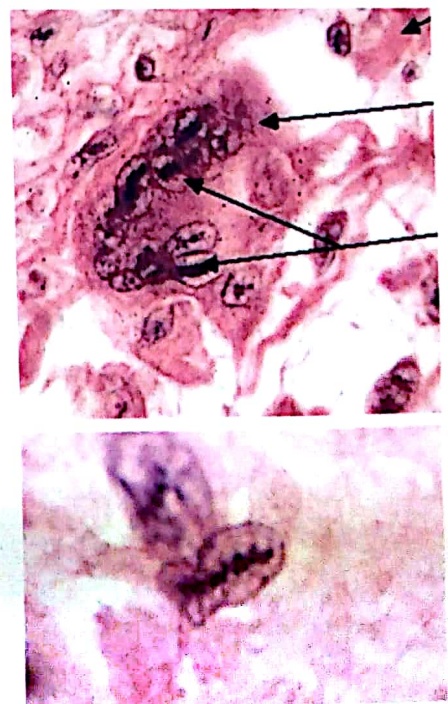
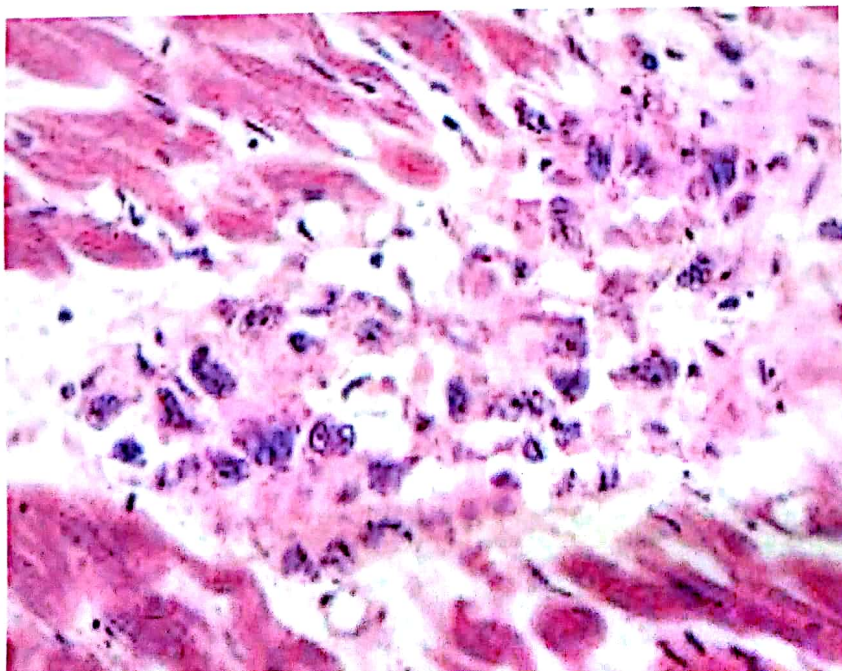
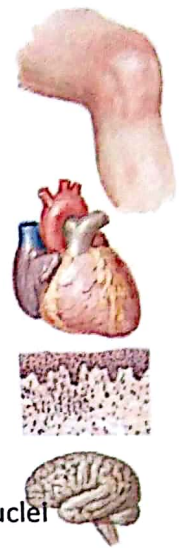
Cross reaction:

- Ab attacks bacterial (M protein) in cardiac muscle → Complement activation → Activation of macrophages

Th → lymphocyte → CD4 T cell attack cardiac muscle → cytokine mediated inflammation

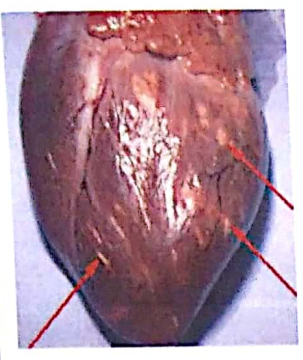
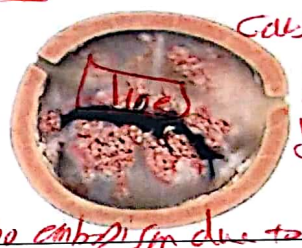
Ashoff's nodule : the pathognomonic lesion

- **Sites:** Heart - Joint - Skin - CNS
- **Gross:** rounded - small (pin head) - pale grey - firm 1-2 mm
- **Micro:**
 - Early:
 - Fibrinoid necrosis (damaged collagen + fibrin deposits)
 - Inflammatory cells (lymphocyte & macrophages)
 - (Anitskow or Caterpillar cell) : Activated macrophage with nuclei containing cylinder beaded chromatin
 - Aschoff's giant cells (multinucleated giant cells, pale acidophilic)
 - Fibroblasts
 - Chronic lesion (in heart only) : Fibrosis + Calcification



Acute Rheumatic heart disease: (Pan-carditis)

common site is posterior wall of left atrium, para vascular and left valves (M, A)

Myocarditis	Pericarditis	Endocarditis
<u>Enlarged & thickened</u> <u>Multiple Aschoff</u> nodules (describe) between muscle fibers  <i>irregularly shaped nodules</i>	<u>Sero-fibrinous inflammation</u>, splitting of 2 layers (<u>Bread & Butter</u>)  Fate: -Resolution -or Fibrosis → Thick white patch (milky patches) → Adhesions <i>vegetation → to mediastinum or to parietal</i>	<u>-Mural endocarditis:</u> Ashoff body + swollen endocardium → Irregular <u>rough patch</u> (<u>Mc Callum patch</u>) → mural thrombosis  <u>-Valve:</u> 75% <u>mitral</u> and <u>aortic</u> - swollen valve, endothelial damage <i>Aschoff nodules, valvulitis</i> <u>VEP</u> = <u>Vegetations:</u> <u>Gross:</u> multiple - Small - pale - adherent on top of the valve along <u>line of closure</u>, <i>leaf</i> <u>micro:</u> platelets + fibrin subendothelial ashoff nodules  <i>cause = Rapid flowing of blood due to faulty valve no embolism due to firmly attached</i>

Chronic Rheumatic Heart disease:

- Aschoff body → fibrosis
- Valve → fibrosis → thick, white & deformed
- Commissural adhesion → Fish mouth or button hole
- Cordae tendinae → thick & short → Funnel shaped valve
- Valve stenosis, or incompetence, or both (double lesion)



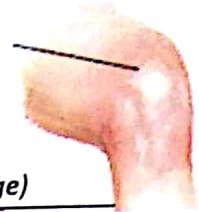
Complications: AFSH

- Arrhythmia (Atrial fibrillation in case of mitral stenosis)
- Fibrosis
- Subacute Bacterial Endocarditis (SBE) → *VIP* → *commensal Bacteria* *Prophylaxis*
- Heart failure (due to chronic valve lesion) → Lung congestion & pulmonary hypertension

N.B. Embolism if occurs (from mural thrombosis - NOT from Rheumatic vegetations)

Rheumatic Arthritis:

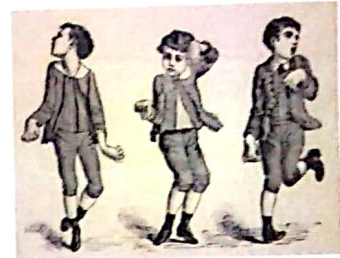
- **Site:** Large joints & muscle , Migratory, fleeting poly-arthritis
- **Morphology:** acute inflammation with effusion *no chronic*
- **Fate:** Self limiting (Acute only → no chronic joint lesion, no joint damage)

**Rheumatic Skin lesion:**

- Subcutaneous nodules: over bony prominence *Wrist, knee*
- Erythema marginatum: red borders and pale center

*احمر**Self limiting***Rheumatic Chorea**

- Rheumatic lesion of Basal ganglia
- Involuntary, purposeless, sudden movement → self limited

موتار *Self limiting*

N.B.

- **Other Rheumatic lesions:-** Arterial nodules, Plurisy, peritonitis
- **Jone's criteria for diagnosis :** 2 major signs or 1 major + 2 minor signs

Major: Carditis, Arthritis, Chorea, Skin nodules, Erythema Minor: Fever, ASO, ESR, Arthralgia

*CRP**التهاب المفاصل*
*بعد التهاب حاد***Endocarditis**

Inflammation of mural & valvular endocardium, with formation of vegetations

Types of Endocarditis = Types of cardiac vegetations:❖ **Non infective** RAT

- Rheumatic endocarditis
- Atypical verrucous endocarditis (Libman-sack endocarditis of SLE) *Systemic lupus erythematosus*
- Thrombotic non bacterial endocarditis (DIC, chronic diseases, metastatic cancer)

Or Associated with any Valve lesion : Valve trauma – Carcinoid syndrome

❖ **Infective** acute – sub - chronic

- Acute bacterial endocarditis (ABE)
- Subacute bacterial endocarditis (SBE)

	Acute infective Endocarditis	Subacute infective endocarditis
Age	> 50 y (lower immunity)	young
Aetiology	Highly virulent bacteria (pyogenic) → septicemia Fungal (with AIDS patients or low immunity) <i>ليس من مميزات</i>	low virulent bacteria (strept. viridans) → bacteremia P.F : - Dental procedures - Diseased valve (e.g. Rheumatic valve – congenital – artificial) - Defective shunt <i>و الشرايين العالقة</i> (ASD, VSD, PDA) - Drug abuse (mainly right sided SBE)
Pathogenesis	Pyogenic bacteria → proliferate on <u>healthy valve</u> → Suppurative inflammation → Suppurative vegetations	weak bacteria → proliferate inside vegetations of diseased valve → non Suppurative inflammation
Morphology	Valve: - suppurative inflammation - ulceration → perforation <i>Sudden death</i> Vegetations: - Large bulky - Yellow - Friable (?) - Micro: - <u>upper zone:</u> platelets - fibrin - <u>middle zone:</u> bacteria - <u>Lower zone:</u> inflammatory cells – Neutrophils & pus cells 	Valve: - non supp. inflammation Vegetations: - Medium sized - less Friable Micro: - <u>upper zone:</u> platelets - fibrin - <u>middle zone:</u> bacteria - <u>Lower zone:</u> inflammatory cells – valve Fibrosis 
Complications	Cardiac: - Valve perforation → acute heart failure - Abscess in myocardium, pericardium Extra-cardiac: - Emboli → pyemia - Toxin → Toxemia	Cardiac: - Valve stenosis -incompetence → Heart failure Extra-cardiac: - Emboli → infarctions - Cerebral, retinal, cardiac, Splenic, renal - <u>very small emboli</u> in vasa vasorum of Arteries - → Mycotic aneurism <i>و</i> - Toxin → Toxemia - Allergic vasculitis (Ag-Ab mediated) & microemboli: - Focal embolic GN (flea bitten kidney) (hematuria, proteinuria & R.F.) - Finger lesions (osler nodule – splinter hemorrhage) <i>بؤس</i> - Petechial hemorrhage in skin & retina <i>بقع</i>

Compare between different types of cardiac vegetations

	Rheumatic	ABE	SBE	Atypical verrucous	Non bacterial Thrombotic
Site	Left valves Upper surface	Left valves Upper surface	Left valves Upper surface	Mitral & tricuspid Upper & <u>lower</u> surface	Any valve Upper surface
Aetiology	Rheumatic fever	Septicemia	Bacteremia	SLE	DIC (e.g. mucoid carcinomas) → ↑ coagulability Debilitating disease (marantic) Damaged valve by trauma → risk
Morphology	describe	describe	describe both side upper lower	Similar to rheumatic + Fibrinoid necrosis	Similar to rheumatic but: Larger (1-5 mm) <u>Loose (embolism)</u>
Fate	Deformity SBE	Pyemia	Embolism	Deformity SBE	Embolism SBE

Carcinoid syndrome:

Serotonin 1381
-GIT carcinoid secretes 5-HT → leads to Flushing & dermatitis - Diarrhea - Asthma

-In case of hepatic metastasis → 5-HT reaches right side of heart

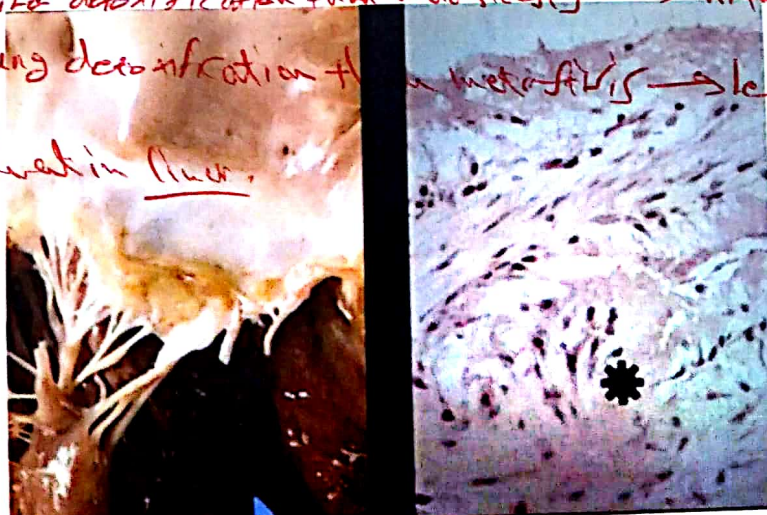
why - thick tricuspid & pulmonary valves -

↑ (smooth muscle + collagen + mucopolysaccharid matrix) - Stenosis

- in case of septal defect or Lung carcinoid → affects left side

Girergerone like desatification than metastasis → Right side
lung desatification than metastasis → left side

Appendix → desatification in liver



Valvular diseases

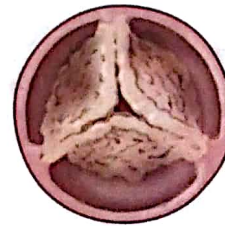
	Mitral stenosis	Mitral incompetence	Aortic stenosis	Aortic incompetence
Causes	- Rheumatic - SBE <i>Sub A. Aortic endo</i> - Congenital	- Rheumatic - SBE - Congenital (e.g. marfan, valve prolapse) - <u>Ruptured papillae or chorda</u>	- Rheumatic - SBE - Congenital - <u>Senile</u> (calcific stenosis)	- Rheumatic - SBE - Congenital (Marfan) - <u>Syphilitic aneurism</u>
Pathological Effects	<u>left atrium</u> dilatation & hypertrophy → <u>Lung congestion</u> → pulmonary hypertension → <u>Right vent dilatation</u> & hypertrophy → <u>Right sided heart failure</u>	<u>left side dilatation & hypertrophy</u> → <u>left sided heart failure</u> → <i>Ax. Ventr.</i> <u>Lung congestion</u> → pulmonary hypertension → <u>Right vent dilatation</u> & hypertrophy → <u>Right sided heart failure</u>	<u>left ventricle dilatation & hypertrophy</u> coronary & cerebral insufficiency Sudden death (VF)	<u>left side dilatation & hypertrophy</u> → <u>left sided heart failure</u> → right sided heart failure

All valvular diseases are complicated by : Thrombosis – SBE & Heart failure

Degenerative valve diseases:

Calcific Aortic stenosis:

- Old age
- Caused by atherosclerosis → calcification
- Valve fibrosis & calcific nodules → valve stenosis
- Lead to IHD & effects of valve stenosis (*enumerate*)



Myxomatous mitral valve prolapse:

- 0.5 to 2.4 % of adults , Asymptomatic

Causes:

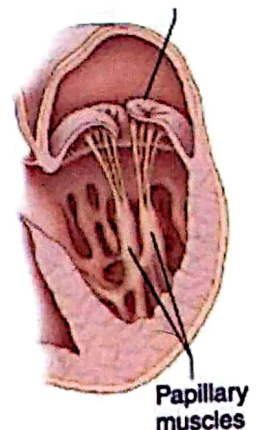
Marfan (Fibrillin-1 mutation) or hemodynamic injury (abnormality in myofibroblast)

Morphology:

Ballooned cusp, thin cordae. Fibrosa layer is thin. Spongiosa layer is myxomatous

Complications: 3%

- Thrombosis & embolism → (infarctions or stroke)
- SBE
- Sudden death (due to VF)



Myocarditis

- **Suppurative**
 - Pyogenic bacteria
 - Pus in interstitial tissue + muscle necrosis
- **Parenchymatous**
 - Toxic (bacterial or chemical) , or parasitic (e.g. Trichinosis)
 - Degenerated muscle + mixed inflammation with eosinophils + necrosis in severe cases
- **Interstitial**
 - Immune response against Viral infection (coxsacki, CMV)
 - Focal muscle necrosis + mixed inflammation with excess lymphocytes
- **Granulomatous**
 - Rheumatic, TB of septum , Sarcoidosis, Syphilis, Fungal (Cryptococcus, Aspergillus, Moniliasis),
 - Microscopic picture of causative granuloma (describe)
 - Idiopathic giant cell myocarditis: rare
 - Affects young adults, idiopathic, some of them are rheumatic, or thymoma patients
 - Ventricular necrosis
 - Chronic inflammatory cells and elongated giant cells

Hypertensive heart

Systemic Hypertension	Pulmonary hypertension (cor pulmonale)
1ry or 2ry	Acute (massive pulmonary emboli) chronic (Idiopathic – Eisenmenger - Lung fibrosis – Emphysema – Lung congestion due to left sided H.F.)
-Increased size >500 g. -Left ventricle is thick >2cm -Nuclear enlargement + fibrosis -Left side dilatation and failure <i>associated with coronary atherosclerosis</i>	- Increased size - Right ventricle is thick -dilatation and failure <i>associated with pulmonary atherosclerosis</i>

Degenerative Heart diseases :

Brown atrophy – Cloudy swelling – Fatty changes – Amyloidosis – Glycogen storage (Von Gierk's disease)

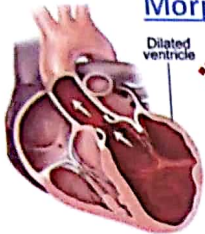
Cardio-myopathy

Pathological changes of myocardium, not caused by ischemia, inflammation, or valve disease

Aetiology

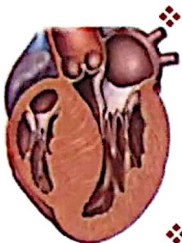
- ❖ 1ry : inherited genetic abnormality
- ❖ 2ry : Sarcoidosis – Amyloidosis – Leukemia – Toxins – Storage diseases (glycogen) ,, "SALTS"

Morphological types



❖ Dilated

- The **most common** type of cardiomyopathy
- Congenital, or 2ry to Toxins (Alcohol, Cobalt Chemotherapy, viral, iron, Beri Beri)
- All chambers are dilated with thin muscle, some fibers are hypertrophied



❖ Hypertrophic

- Congenital autosomal dominant (B-myosin, C protein, Troponin T)
- Asymmetrical hypertrophy of left ventricle & ventricular septum → **banana** shaped distorted ventricle → Diastolic dysfunction & Mitral regurge & H.F.
- Haphazard myocytes & fibrous tissue



❖ Restrictive

- Rare type
- Idiopathic fibrosis, or 2ry to Amyloidosis, endomyocardial fibrosis, Loffler's dis
- Thickening of endocardium → limitation of diastolic filling
- Endomyocardial fibrosis : caused by starvation or eosinophilia
- Loffler's: endocardial fibrosis due to eosinophilia

Ischemic Heart diseases (IHD)

Angina – Chronic IHD - Myocardial infarction – Sudden cardiac death

causes:

- <u>A</u>ortic aneurism (syphilitic, Dissecting) - <u>A</u>ortic calcific stenosis - <u>A</u>ortic vlave stenosis & Coarctation	- <u>C</u>oronary <u>A</u>therosclerosis - <u>C</u>oronary Thrombosis – embolism - <u>C</u>oronary spasm - <u>V</u>alve diseses - <u>V</u>asculitis (PAN)	- <u>H</u>ypotension (shock) - <u>H</u>ypertension - <u>H</u>ypoxemia (Anemia, CO)
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	Angina	Chronic IHD
Causes	1-Stable: crushing or squeezing, induced by <u>effort</u> <u>occlusion of > 70%</u> of lumen 2-Prinzmetal: <u>Vasospasm</u> giving pain <u>during rest</u> 3-Unstable (crescendo): <u>increasing pain</u> intensity, caused by low effort + <u>occlusion of >90%</u>	Progressive H.F. due to old infarction
morphology	No morphological changes until infarction	Gross: dilated, gr. White Micro: fibrosis + hypertrophy + degenerated vacuolated cells

Myocardial Infarction

Coagulative necrosis of myocardium due to ischemia

Incidence: Most common cause of *disease-induced death*. Male > female, >45 y

P.F.: causes of IHD, mainly **Complicated atherosclerosis**



Sites:

- Left anterior coronary occlusion (40-50%) → infarction of anterior wall of left ventricle, ant 2/3 of septum & apex
- Right coronary occlusion (30-40%) → infarction of posterior wall of left ventricle, right ventricle, post 1/3 of septum.
- Left circumflex occlusion (15-20%) → infarction of lateral wall of left ventricle

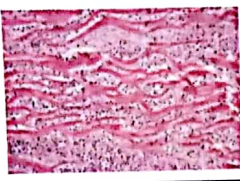
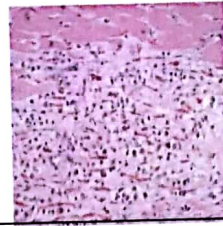
Pathogenesis:

- Coronary ischemia by stenosis (atherosclerosis) or occlusion (thrombosis, spasm) → Hypoxia → lack ATP → Loss of contractility (in one minute) + Lactic acidosis
- **Necrosis** → release of enzymes **CK, LDH**, myocardial protein **Troponin I (c-Tn-i)**
- Ischemia 20 – 40 min is irreversible, while during 3-6 hours intervention can prevent extension of infarction.

Patterns:

Sub-endocardial	Transmural	Microscopic
-1/3 thickness, multifocal -stenosis but not occluded: caused by effort, or occurs during night -Arrhythmia	-Full thickness -caused by occlusion – more complications	-small areas -small vessel occlusion (e.g. vasculitis) -less complicated

Morphology::

0.5 -2 hours reversible	4 hours – 1 st day	1 st – 3 rd day	1 st – 2 nd week	2 nd week-2 nd month
Gross: none	Dark, mottled	Pale yellow	Pale yellow Red margins	Grayish white Firm
Micro: Wavy fibers E/M: glycogen loss ,mitochondria swollen 	Coagulative necrosis -eosinophilic fibers - pyknosis - neutrophils 	-no <u>striations</u> -no <u>neuclei</u> -neutrophils - <u>MQ</u> appear 3-7 days 	Granulation tissue at <u>margin</u> (describe) 	Collagen scar 

Complications: CHAMP

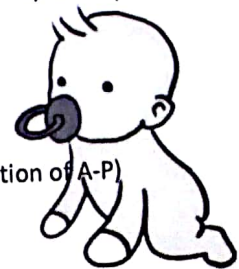
- **C**ongestive heart failure
- **H**emopericardium (Tamponade) : due to ruptured pap. muscle → acute H.F.
- **A**neurismal dilatation (due to fibrosis) → thrombosis & embolism
- **A**rrhythmia (ischemia of conduction system)
- **M**yocardial rupture (left vent, papillary muscle, or vent septum) during 7 days
(muscle is weak and infiltrated by inflam. cells , before granulation tissue)
- **P**ericarditis

Tumors of the Heart:

Myxoma of left atrium(the most common) - Rhabdomyoma – Lipoma – Fibroma- Angiosarcoma (the most common 1ry malignant) – metastatic (e.g.leukemia, lymphoma, Lung carcinoma , melanoma)

Congenital Heart diseases

- **Incidence:** 10%of heart diseases in children, starts in 5th to 8th gestational week
- **Causes:** Teratogenic Drugs– Trisomies (e.g. Trisomy 21) – D.M.- viral (Rubella, coxaci)-
- **Types:**
 - **Stenotic** diseases : Aortic coarctation
 - **Shunt** diseases :
 - **Non cyanotic** left→right shunts (ASD, VSD, PDA)
 - **Cyanotic** right→left shunts (Fallot tetralogy, Transposition of A-P)



Aortic Coarctation:

Focal stenosis in the wall of Aorta, 2 types:

Pre-ductal	Post-ductal
-<u>Proximal</u> stenosis (before PDA) -Associated with <u>PDA</u> -<u>Fatal</u> in early life (infantile)	-<u>Distal</u> stenosis -No PDA -Not fatal (Adult) , complicated with upper limb <u>hypertension</u>, lower body <u>hypotension</u>

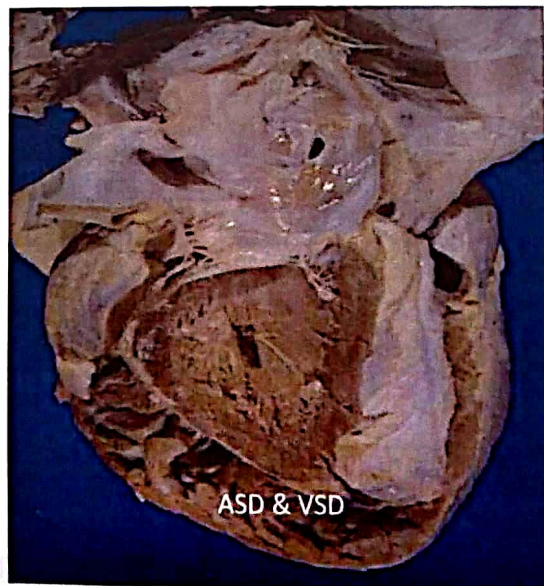
Shunt diseases:

Left → right shunt (non cyanotic)	Right → left shunt (cyanotic)
-ASD patent foramen ovale, (the most common) -VSD <u>Large defect</u>– <u>Small defect</u> of Roger -PDA	-Tetralogy of Fallot -Transposition of great vessels (A-P)
<u>Effects:</u> -Right s. dilation → rt. S.H.F. (congestive H.F.) -SBE - Reversal of shunt (Eisenmenger) → Cyanotic	<u>Effects:</u> -Left s. H.F. -SBE -Hypoxia → Cyanosis & polycythemia & clubbing of fingers

PDA : In normal infants DA is closed after 8th week of birth due to low Pg E2, & hypoxia.

Tetralogy of Fallot : P.S. – Right ventr hypertrophy – Overriding of Aorta – VSD PROV

Transposition : Aorta arises from right, and pulmonary arises from left.



Heart Failure

Maintained impairment of cardiac function, with insufficient cardiac output

Acute : pulmonary embolism – Hemopericardium (tamponade) – Myocardia disease (?)

Chronic:

Left Sided H.F.	Right sided H.F.
Causes: CAMPS -Congenital (right → left shunts enumerate?) -Aortic diseases -M.I (mitral incompetence- myocardial infarction) -Pericardial disease (pericarditis – hemopericardium) -Systemic hypertension	Causes: -Congenital (left→right shunts enumerate?) -P.S. -M.S -Pericardial disease (pericarditis – hemopericardium) -Pulmonary hypertension (lung fibrosis – emphysema – lung congestion due to left sided HF)
Effects: -Lung congestion → brown induration? -Right sided heart failure	Effects: Chronic generalized venous congestion ?

Congestive Heart Failure = Left sided heart failure + Right S. H. F.

Pericarditis

Acute

- **Serous**: infection (viral)– immunological (Rhumatic, SLE) – idiopathic
- **Fibrinous** (same causes + uremia) → heal by organization → adhesion
- **Suppurative** (direct, blood, lymphatic) → marked adhesions
- **Hemorrhagic** fibrinous or suppurative + blood (TB, tumors, Trauma, blood diseases) → marked adhesions

Chronic

- **Adhesive** : adhesions obliterate pericardial sac → limited complications
- **Constrictive**: fibrous calcific scar around the heart (defective diastole) and may obliterate orifices of vena cava → generalized congestion
- **Tuberculous** (fibrinous + GT and fibrosis) → adhesive or constrictive

Hemopericardium (tamponade) is caused by: General causes of bleeding– Local causes (injury – tumor – ruptured cardiac or aortic aneurism). Effect → limited diastole → HF

Pneumopericardium: Air in pericardium (Lung trauma – or gas forming organisms)

MCQ

Q1: Regarding rheumatic vegetation all are true except:

- a) Appeared on the ventricular surface of the mitral valve.
- b) Multiple beaded thrombi.
- c) The thrombi are pin head size.
- d) Never give embolic manifestation.

Q2: The type of necrosis that develops in Aschoff nodule is:

- a) Coagulative necrosis
- b) Fibrinoid necrosis
- c) Liquifactive necrosis
- d) Zenker necrosis

Q3: Mycotic aneurysm that may develop in cerebral vessels due to low grade infected emboli is a complication of:

- a) Acute bacterial endocarditis
- b) Acute rheumatic valvulus
- c) Subacute bacterial endocarditis
- d) Libman-Sacks endocarditis

Q4: Male patient 23 years old developed sudden severe chest pain, with no any relevant past history, followed by death, postmortem examination revealed enlarged congested heart with friable necrotic foci, microscopic examination revealed inflamed myocardium with mononuclear cellular infiltrate rich in giant cells and areas of necrosis, the most likely diagnosis of this case is

- a) Acute myocardial infarction
- b) Giant cell myocarditis.
- c) Acute Rheumatic myocarditis
- d) Acute bacterial endocarditis

Q5: in a case of myocardial infarction, well developed phagocytosis with prominent granulation tissue formation. Appeared at:

- a) 2-24 h
- b) 1-3 days
- c) 4 -7 days
- d) 7-14 days

Q6: Infarction in the posterior wall of left ventricle and posterior part of interventricular septum is caused by occlusion of:

- a) Descending branch of left coronary
- b) Right coronary
- c) Left circumflex
- d) Left coronary

Q7: Pericarditis that developed 2 to 6 weeks after myocardial infarction is believed to be

- a) Infectious
- b) Immunological mediated
- c) Traumatic
- d) Toxic

Q8: Regarding tuberculous pericarditis,

- a) Inflammatory exudate rich in neutrophil-, is detected
- b) It can be hemorrhagic but complete resolution is the usual fate
- c) It is a primary tuberculous infection.
- d) None of the above

Q9: Cardiac tamponade is

- a) Aneurysmal dilatation of myocardial infarction
- b) Mural thrombosis with embolization
- c) Cardiac perforation with accumulation of blood in pericardial sac that interfere with normal heart contraction.
- d) None of the above

Q10: Fallot's tetralogy includes all except

- a) Hypertrophy of left ventricle
- b) Pulmonary stenosis
- c) Shift of aorta to right side
- d) High ventricular septal defect

Q11: Regarding coarctation of the aorta, it is accompanied with

- a) Right to left shunt
- b) Left to right shunt
- c) No shunt
- d) None of the above

Q12: Monckeberg's medial calcific sclerosis shows

- a) Calcification in vessel wall with narrowing of the lumen
- b) Strong relationship to atherosclerosis
- c) No clinical manifestations
- d) Affection of young people

Q13: The most common cause of death in essential benign hypertension is

- a) Congestive heart failure
- b) Cerebral vascular accident (hemorrhage)
- c) Renal failure
- d) None of the above

Q14: Fibrinoid necrosis is a common pathological feature seen in

- a) Atherosclerosis
- b) Benign hypertension
- c) Malignant hypertension
- d) None of the above

Q15: Regarding Buerger's disease

- a) It commonly affects the large vessel
- b) The vessel wall shows mixed acute and chronic inflammatory cells
- c) It is limited to the arterial wall
- d) It has no clinical significance

Q16: Hepatitis B virus antigens incorporated in which of the following diseases

- a) Giant cell arteritis
- b) Rheumatic arteritis
- c) Buerger's disease
- d) Polyarteritis nodosa

Q17: The vegetations that give aseptic emboli occur with

- a) Non-bacterial thrombotic endocarditis.
- b) Acute Rheumatic endocarditis
- c) Acute bacterial endocarditis

Q18: Regarding Von Gierke's disease, the cardiac muscle fibers are enlarged and vacuolated due to

- a) Accumulation of lipid
- b) Accumulation of amyloid material

- c) Accumulation of glycogen
- d) None of the above

Q19: Concerning suppurative pericarditis, all are true except

- a) It is yellow creamy in appearance
- b) It showed increase neutrophils and pus cells
- c) It ends by resolution
- d) It may be haemorrhagic

Q20: Secondary hypertension can be caused by all except

- a) Conn's disease
- b) Cushing syndrome
- c) Pheochromocytoma
- d) None of the above

Q21: Dissecting Aortic aneurysm can be seen in

- a) Myxomatous degeneration of aortic media
- b) Polyarteritis nodosa
- c) Atherosclerosis
- d) None of the above

Q22: Glomangioma (Glomus tumour) is

- a) Malignant vascular neoplasm
- b) Benign vascular neoplasm
- c) Hamartoma
- d) None of the above.

Q23: Phlebothrombosis means

- a) Thrombosis of non-inflammatory origin
- b) Thrombosis initiated by inflammation
- c) Thrombosis with or without inflammation
- d) None of the above

Q24: If a trauma leads to fistulous communication between the wall of artery and vein via a hematoma, the patient is considered to have:

- a) Vascular malformation
- b) False aneurysm
- c) Vascular tumour
- d) None of the above

Q25: Hyalinosis and elastosis are diagnostic findings in the arterial wall affected by:

- a) Atherosclerosis
- b) Benign hypertension
- c) Malignant hypertension
- d) None of the above.

Q26: A male patient 10 years old presented of fever and malaise, clinical examination revealed inflamed knee with skin rashes. On auscultation, murmur is heard over his mitral valve. History is taken and is suggestive for the diagnosis of his condition. What is the most important test that should be done to confirm the diagnosis?

- a) ECG
- b) B- Antistreptolysin O titre
- c) C reactive protein
- d) ESR

Case 1

A male patient 65 years old heavy smoker, obese and hypertensive, died from embolic brain infarction. Postmortem examination revealed rough aortic intima with confluent ulcerated yellowish patches.

- 1- What is the aortic lesion that present in this patient?
- 2- What are the risk factors for his condition?
- 3- What is the pathogenesis of his disease?
- 4- List two complications for this disease?

Case 2

During postmortem examination of a cadaver who died in car accident, the heart showed thickened wall about 2.5 cm in thickness, kidneys were contracted with fine granularity on their outer surface, otherwise no any other gross abnormality appeared in the examined internal organs.

- 1- What do you expect the patient had before death?
- 2- What do you expect to see in the small vessels on microscopic examination?
- 3- How is this disease classified, give examples.

Case 3

A 12 years old boy was brought to the physician with a sore throat and fever 3 weeks ago. On examination, the child is afebrile but audible murmur on the mitral valve is found. The child is admitted in the hospital, over the next 2 days the child develops manifestations of left ventricular failure.

- 1- What is your diagnosis?
- 2- What is the pathogenesis of this disease?
- 3- List two complications for this disease.
- 4- In your opinion, what is the cause of left ventricular failure in this patient?

Case 4

Male patient 55 years old with hyperlipidemia and hypertension developed a severe attack of chest pain that was not responding to repeated sublingual nitroglycerine treatment. The patient died. Postmortem examination revealed yellow soft wall of the wall of the left ventricle, apex and the anterior part of interventricular septum. Dissection of one of the coronaries revealed thrombotic occlusion.

- 1- What is the cause of death in this patient?
- 2- What is the occluded coronary?
- 3- According to the gross appearance of the left ventricular wall how long did it take this lesion to occur and what do you expect to see microscopically?
- 4- Enumerate four complications that would develop if this patient was still alive?